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Review Article

Focal Atrial Tachycardia

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Over the past decade, most of the electrophysiologic mechanisms in patients with focal atrial tachycardias (ATs) have been well studied. Recently, advanced mapping systems have revealed the precise propagation, and the relationship between non-radial propagation pattern and substrate property of focal AT. Non-radial activation pattern of focal AT may mimic macrorentrant AT and may lead to inappropriate ablation strategy. In this review, we will focus on the current state of mapping and ablation techniques, safety, and efficacy associated with the catheter ablation for focal AT.
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Key words: Focal atrial tachycardia, Catheter ablation

Introduction

By the classic definition, focal atrial tachycardia (AT) is defined as supraventricular tachycardia originating from a discrete area (origin) from which it centrifugally spreads out with regular atrial rhythm.[1)] The atrial activation rate is less than 250 beats/min and the discrete P-waves are separated by isoelectric intervals, and it may present as either a paroxysmal, incessant, or persistent tachycardia. Radiofrequency (RF) catheter ablation has become a first-line therapy for the management of drug refractory focal AT according to the ACC/AHA/ESC guidelines.[2)]

Epidemiology

Focal AT is a relatively uncommon supraventricular tachycardia. The prevalence in asymptomatic subjects with this tachycardia was reported as 0.34% and in symptomatic patients as 0.46%.[3)] Focal AT can be diagnosed in children or adults without any structural heart disease, but in the elderly it is often associated with cardiovascular disease.[4–17)] The persistent or incessant form of focal AT can cause congestive heart failure due to tachycardia-induced cardiomyopathy, which is more frequent in pediatric patients than in adults.[18,19)]

Pathophysiology

Mechanisms

The mechanisms of focal AT can be abnormal automaticity, triggered activity, and microreentry.[12,20–23)] Automatic AT can be identified by the following characteristics: 1) AT onset is followed by a gradual acceleration (*warm-up*) and termination

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follows slowing (*cool-down*); 2) AT initiates spontaneously or by isoproterenol loading; 3) AT cannot be initiated, entrained or terminated by programmed electrical stimulation; 4) AT can be transiently suppressed by overdrive atrial pacing.[12] AT related with triggered activity can be identified by the following characteristics:[12,21,22] 1) AT can be initiated or terminated by programmed stimulation, and its initiation is cycle length-dependent; 2) pacing can not entrain tachycardia, but can cause overdrive suppression or termination; 3) early afterdepolarization can be found before full repolarization, or delayed afterdepolarization can be found after completion of repolarization (phase 4) just before the onset of the AT during the monophasic action potential recoding at the earliest activation site. AT related with reentry mechanism can be identified by the following characteristics: 1) AT can be reproducibly initiated and terminated by programmed stimulation; 2) AT can be entrained by pacing; 3) AT can show inverse relationship between the coupling interval of first AT beat and atrial premature beat. Because of triggered activity and reentry possess a significant overlap in the electrophysiologic characteristics, there is no definitive diagnostic criteria to differentiate these two mechanisms. Further evaluation is needed to assess whether the likely arrhythmia mechanism is related to clinical behavior of the arrhythmia and its response to treatment.

Pathohistology

Previous studies of surgical treatment in patients with focal AT demonstrated the atrial specimen resected from the area with ATs showed a slow-response or depressed fast response action potential with spontaneous depolarization.[24,25] Myocardial fibrosis, fatty infiltration, and hypertrophy were seen in the atria around AT origin.

Pharmacology

The pharmacologic response likely does depends on the AT mechanism. Adenosine, verapamil and propranolol can terminate most triggered activity related focal AT.[12,23,26] Recently, lidocaine-sensitive AT has been reported, but the mechanism is still unknown.[27] Termination of AT by adenosine suggests a focal AT. On the other hands, failure of adenosine to terminate AT often suggests macrorrentrant AT.[23] Failure of adenosine to terminate AT accompanied with AV block can facilitate identification of P-waves, and is diagnostically useful to exclude AV reentrant tachycardia and makes AV nodal reentrant tachycardia unlikely as a tachycardia mechanism.

Diagnosis

Focal AT can be diagnosed if the following criteria are observed: 1) the endocardial activation sequence during AT is different from that during sinus rhythm and ventricular pacing; 2) atrioventricular nodal block does not affect the AT; 3) changes in PR and RP intervals follow changes in the AT rate.[12,28,29] If focal AT originate from the area near a valve annulus or the AV node, pacing maneuvers are useful to exclude atrioventricular nodal reentrant tachycardia (AVNRT) or atrioventricular reentrant tachycardia (AVRT) using accessory pathway.[12,28,29] Our first step is to insert ventricular stimulation during tachycardia (**Figure 1**). AT can be excluded if a ventricular pacing reproducibly terminates tachycardia without conduction to the atrium. If a premature ventricular beat advances atrial activation when the His-bundle is refractory, the presence of an AV accessory pathway is confirmed, although it does not prove that the tachycardia uses the pathway. Second, trains of ventricular stimuli at a cycle length just shorter than the tachycardia cycle length are also helpful. If the ventricle is dissociated from the tachycardia, AVRT using an accessory pathway is excluded. If ventricular pacing produces V-A conduction and a different endocardial activation sequence from that of the tachycardia, AT is likely. If ventricular pacing produces VA conduction without interrupting tachycardia, the pattern of resumption of tachycardia distinguishes AT from AVRT or AVNRT.[29] When the last atrial electrogram (A) that is advanced by the ventricular stimulus is followed by a ventricular (V) or His deflection (A-V response) AVNRT or AVRT is the mechanism. When the last stimulated A is followed by another A that continues tachycardia (A-A-V response) (**Figure 1**) AT is the diagnosis.[29] A third maneuver is atrial stimulation at a shorter pacing interval than the tachycardia cycle length. The faster pacing rate typically induces some conduction delay at the AV node which resolves when pacing is stopped, and then the tachycardia rate returns to the baseline tachycardia rate. A fixed VA interval (variation range less than 10 ms) as the tachycardia cycle length varies, indicates that AVNRT or AVRT is likely. Tachycardia continues after the atrial pacing is stopped, a VA interval of the return cycle similar to that of the tachycardia (variation range less than 10 ms) does not favor AT. In contrast, a variable change in the VA interval

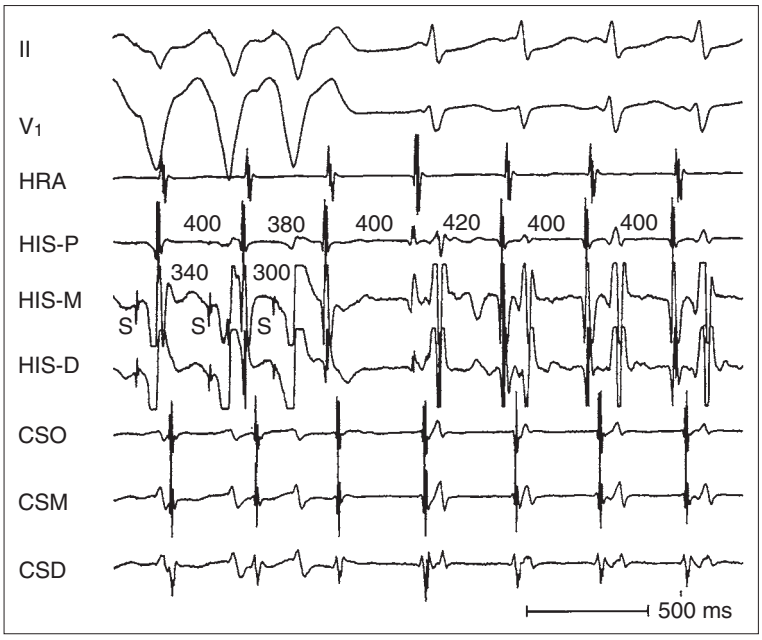

Figure 1 Intracardiac tracings indicate insertion of three ventricular stimuli with coupling intervals 340 and 300 ms during AT.
The last stimulus advanced atrial activation to A-A interval of 380 ms. The AT continued after a following atrial electrogram sequence (A-A-V response), consistent with AT.
HRA = high right atrium; HIS = His bundle area; CS = coronary sinus; O/P/M/D = ostium, proximal, middle, distal. (adapted from reference 73, Hsieh and Chen).

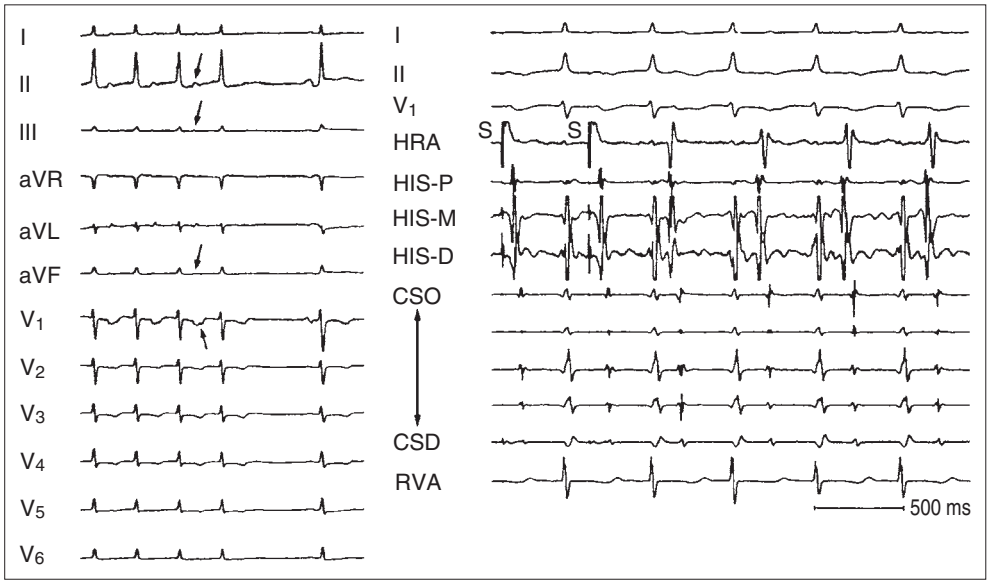

Figure 2 Anteroseptal AT.
Left panel: 12-lead ECG recordings during spontaneous termination of AT. Arrows indicate the last AT beat, which is biphasic in lead V1 and positive in inferior leads. Right panel: AT initiation by right atrial pacing (S). Earliest atrial activation is recorded from the His-bundle area. The VA interval after atrial pacing is variable, longer immediately after pacing, suggesting atrial tachycardia rather than AV nodal reentry. Abbreviations are the same as for Figure 1. RVA = right ventricular apex. (adapted from reference 36, Chen et al.).

suggests AT (**Figure 2**).[29] Finally, AV block when pacing the atrium at the tachycardia cycle length immediately after tachycardia termination suggests that AV nodal conduction is required for tachycardia and AT is unlikely.

Two types of AT involving the sinus node exist. First, sinus nodal reentrant tachycardia is defined as a tachycardia originating from the sinus node complex and showing reentry mechanism.[30] The P-wave morphology and the endocardial activation sequence during the tachycardia are identical or similar to those during sinus rhythm. Whether this

AT truly originates from the sinus node or adjacent tissue is still controversial.

Second, inappropriate sinus tachycardia (IST) also originates from the sinus node complex without property of reentry mechanism. This tachycardia is characterized by an increased heart rate in resting (more than 100 beats/min) or under loading minimal exertion or body posture change.[31–33] Abnormal function of autonomic nervous system or a primary abnormality of the sinus node itself have been suggested as an underlying mechanism of this tachycardia. For its diagnosis, secondary causes of the sinus tachycardia including endocrinological disorders, and other tachycardia originating from upper portion of crista terminalis or superior vena cava (SVC) should be excluded. However, change in autonomic tone may shift the earliest activation site along the crista terminalis. Therefore, the distinction between IST and focal AT from the crista terminalis or SVC tachycardia is practically difficult.

Anatomy

Focal ATs tend to have a characteristic anatomic distribution associated with specific structures showing the anisotropy or poor intercellular coupling, and these structures may play possible role as an arrhythmogenic substrates (**Figure 2–6**).[11,34] In the right atrium, common locations of AT origins are the crista terminalis, right-side of the interatrial septum including the vicinity of the AV node, right atrial appendage, tricuspid annulus, coronary sinus ostium, and the SVC.[11,26,34–42] In the left atrium, AT origin tend to cluster the pulmonary vein ostium, the left atrial appendage, left-side of the interatrial septum, the coronary sinus body, mitral annulus, the ligament of Marshall, or a left-sided SVC.[43–51]

Mapping

12-Lead Surface ECG

The assessment of the 12-lead ECG P-wave polarity can facilitate to estimate an approximate location of the AT (**Figures 2–5**). In general, focal AT is characterized by discrete P-waves separated by isoelectric intervals in all ECG leads. However, a continuous undulating without an isoelectric baseline can be seen in some unique focal ATs. Furthermore, the merging of P and preceding T waves during AT often obscures the true morphology of the P-waves. In such ATs, the insertion of premature ventricular stimuli to advance the ventricular depolarization (**Figures 4** and **7**) or creation of transient AV block with adenosine loading is useful to record the pure P-waves during the AT.

Leads aVL and V1 are the best leads to distinguish right atrial from left atrial foci.[53,54] A negative or isoelectric P-wave in lead aVL and positive P-wave

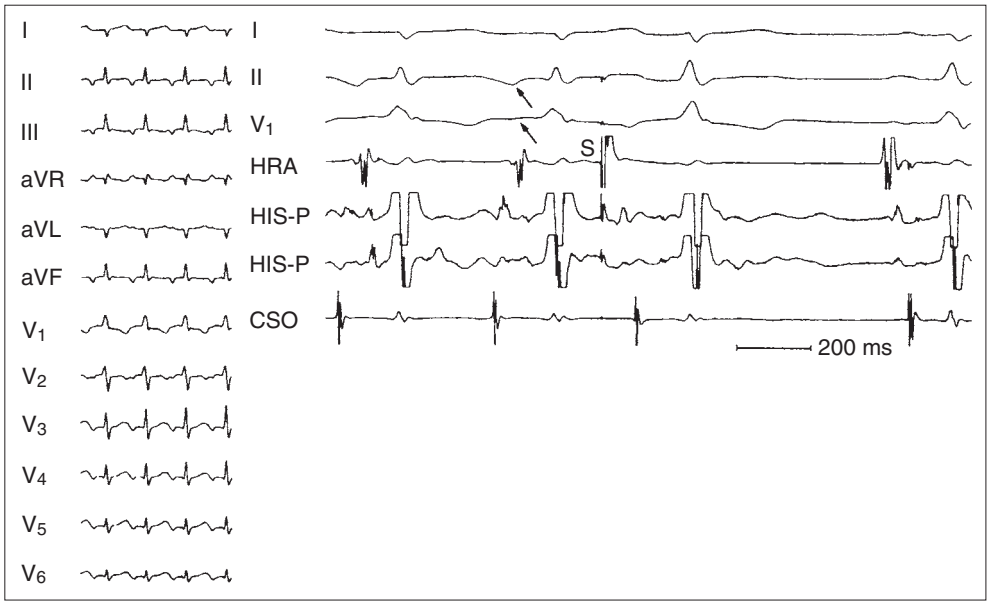

Figure 3 Midseptal AT.
Left panel: 12-lead ECG recordings during AT. Right panel: AT termination by a premature stimulus (S), followed by a sinus beat with biphasic P-waves (positive-negative) in V1. Comparison of the last AT beat (arrow) and the sinus beat shows that this midseptal AT exhibits a biphasic P-wave (negative-positive) in lead V1 and a negative P-wave in lead II. (adapted from reference 36, Chen et al.).

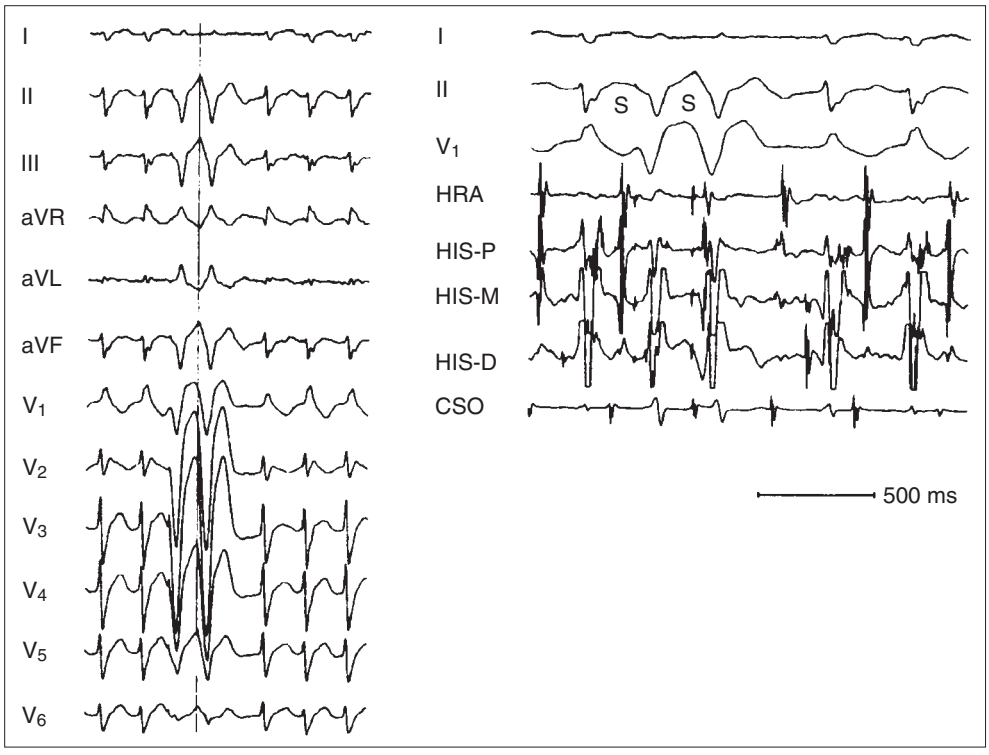

Figure 4 Posteroseptal AT.
Left panel: the 12-lead ECG during AT. Two premature ventricular stimuli were delivered during AT to expose the P-wave. The P-wave polarity is positive in lead V1 and negative in inferior leads.
Right panel: Intracardiac recordings during AT. Earliest activation during AT is at the CSos.
HRA = high right atrium; HIS-P/HIS-M/HIS-D = proximal, middle, distal part of His-bundle recording; CSO = coronary sinus ostium. (adapted from reference 36, Chen et al.).

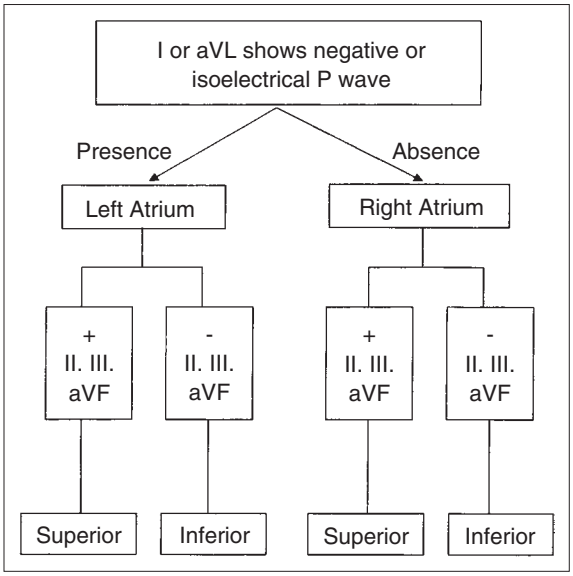

Figure 5 An algorithm demostrates a scheme to estimate the focal AT origin based on the P-wave polarity of the 12-lead ECG recordings.
A negative or isoelectric P-wave in lead I or aVL suggests a left atrial focus. Positive P-waves in inferior leads indicate superior (cranial) origins. (adapted from reference 73, Hsieh and Chen).

in lead V1 suggest a left atrial foci. Furthermore, a positive or biphasic P-wave in lead aVL and negative or biphasic P-wave in lead V1 indicated a right atrial origin. In addition, positive P-waves in the inferior leads suggest a superior or anterior origin, and biphasic or negative P-waves in the inferior leads indicate a posterior or inferior origin. Other proposed criteria of the P-wave polarity predicting the AT focus are suggested as follows: 1) a negative P-wave in lead aVR usually indicates a crista terminalis origin;[55] 2) a negative P-wave polarity in the anterior precordial and inferior leads suggests AT from the inferoanterior portion of the tricuspid annulus;[34] 3) a combination of a negative P-wave in V6, positive P-wave in V1 and negative P-waves in all inferior leads suggests a posteroseptal AT near the coronary sinus ostium;[36] 4) a monophasic positive P-wave in lead V1, observed with ATs originating from the vicinity of the AV node, indicates a left-sided interatrial septal origin;[37] 5) an almost identical P-wave morphology during the AT to sinus rhythm indicates an AT from upper portion of the crista terminalis or sinus tachycardias.[11,30–33]

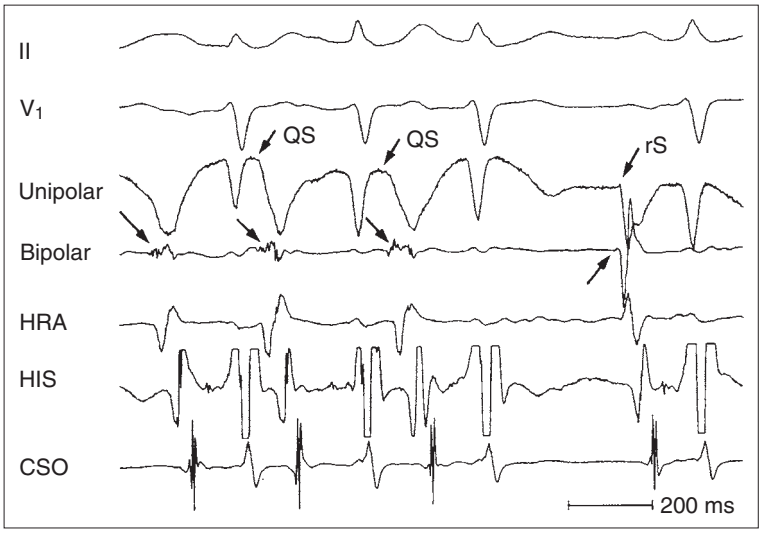

Figure 6 Simultaneous recordings of unipolar and bipolar recordings from the successful ablation site of AT (first three beats) and sinus rhythm after spontaneous AT termination (last beat).
Arrows indicate the onset of the atrial electrograms recorded from the ablation site. Bipolar recordings from the ablation catheter shows fractionated electrograms during AT and discrete electrograms during sinus rhythm. The unipolar recording demonstrates a QS pattern during AT, consistent with wavefronts moving away from the recording site, and an rS pattern during sinus rhythm.
HRA = high right atrium; HIS = His bundle area; CSO = coronary sinus ostium. (adapted from reference 73, Hsieh and Chen).

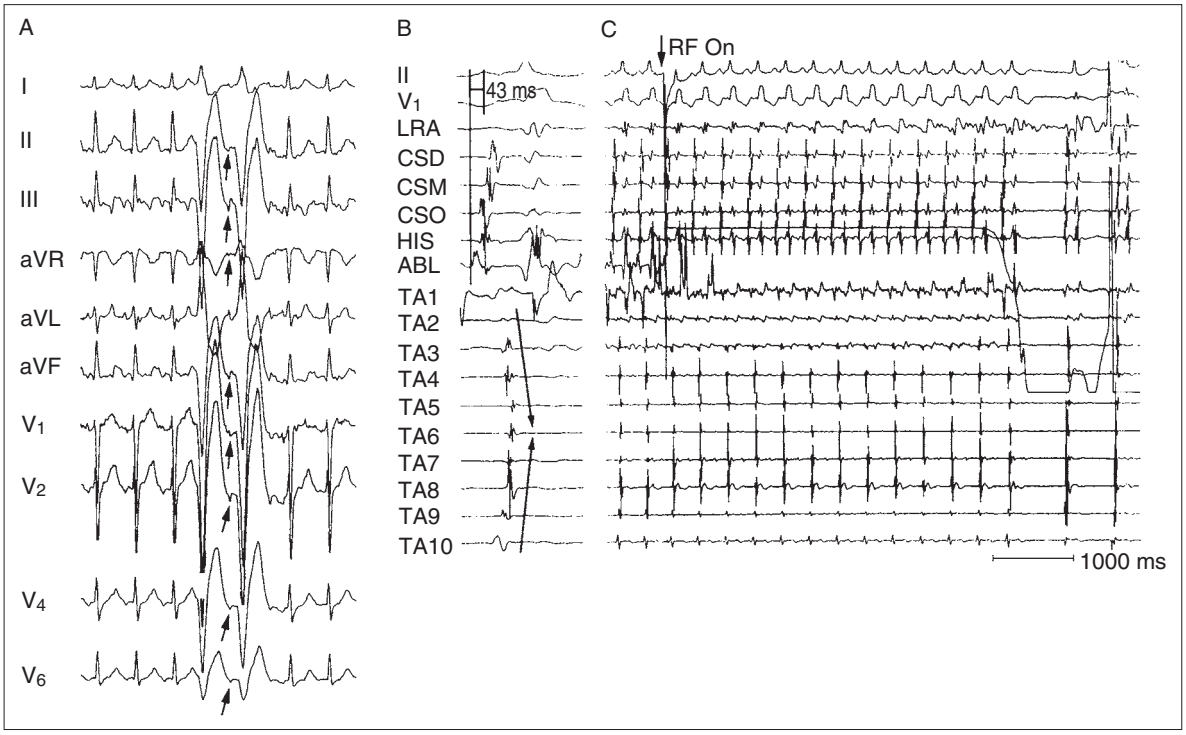

Figure 7 AT originating from the tricuspid annulus.
(A) 12-lead ECG indicates two premature ventricular stimuli during AT unmask the P-wave morphology (arrows). P-wave polarities are negative in the precordial leads and inferior leads, and positive in lead I and aVL. The P-wave polarity favors the origin of AT from the tricuspid annulus. (B) The electrogram at the successful ablation site during AT shows a small A wave and a large V wave indicating origin near the tricuspid annulus. The atrial activation timing at the ablation site precedes 43 ms before the onset of surface P-wave. (C) Abrupt termination of AT during RF energy application were observed.
LRA = low right atrium; CS = coronary sinus; D/M/O = distal, middle, ostium; HIS = His bundle area; ABL = ablation catheter; TA = tricuspid annulus. (adapted from reference 73, Hsieh and Chen).

Usually, an electrically silent period exists within the atrium with an isoelectric baseline between each P-wave on the surface ECG and absence of atrial electrograms on the endocardial recordings. However, some unique ECG patterns of focal AT are reported. If the AT cycle length is enough short and

intraatrial conduction delay exists, atrial activation time during the AT may become prolonged such that the P-wave and intraatrial electrogram occupy a large proportion of the AT cycle length. In this unusual condition, the surface ECG P-wave morphology may demonstrate an atrial flutter like pattern with continuous undulation without an isoelectric baseline.[56] Rapid AT can cause a focal atrial fibrillation. A very fast focal discharge with variable exit block from the arrhythmogenic focus or non-uniform centrifugal spreads from the focus with a fibrillatory conduction pattern in the reminder atria can rise to the electrocardiographic pattern of atrial fibrillation.[52]

Endocardial Activation Sequential Mapping

First, endocardial activation mapping starts with routine catheters positions, such as the high right atrium, coronary sinus and His-bundle region for estimation of the AT focus based on the atrial activation pattern. Second, further detailed mapping is performed by an ablation catheter positioned around the region of interest to identify the earliest activation timing relative to the P-wave onset or atrial electrogram deflection recorded from electrodes at the fixed positions such as the coronary sinus ostium or high right atrium. Another simple method is the "two catheter technique". Two roving ablation catheters are alternately moved to search the site of early atrial activation. The earliest activation timing at the successful ablation site is usually at least 30 ms before the P-wave onset, but may vary greatly among the patients.[4–17] Due to the conventional endocardial mapping technique has limited resolution, several specialized mapping catheters has been developed. A self-expandable multielectrode basket catheter (Constellation, Boston Scientific) can also facilitate for simultaneous global atrial endocardial mapping to rapidly identify the AT origin and to navigate the ablation catheter inside the basket catheter (Astronomer System, Boston Scientific).[13] Limitations of this catheter are incomplete endocardial coverage of the atrium, especially in the appendage and isthmus, and a risk of systemic thromboembolism when mapping is performed in the left atrium. Sanders et al. developed novel high-density self-expandable catheter with 5 radiating spine (PentaRay, Biosense-Webster) and it can be useful for vector mapping and localize complex ATs.[57]

Atrial anatomy is complex and neighboring structures can lead misinterpretation of electrical signals. The right superior pulmonary vein locates close proximity to the right atrium, so that electrical signals from both structures can be recorded simultaneously and AT from either site can have a similar P-wave morphology. Therefore, when the early activation site locate around the upper portion of the crista terminalis or right atrium-SVC junction, the possibility of a right upper pulmonary vein tachycardia should be considered.[11] If far-field pulmonary vein potentials precedes the high posteromedial right atrial direct potentials, it indicates a AT from right pulmonary vein.

Diagnosis of septal ATs can be challenging (**Figures 2–4**). Focal AT can originate from either side of the interatrial septum near the AV node.[37] Careful RF ablation in this area would avoid a potential risk of AV block. Focal AT from right sided interatial septum can be excluded if the following observations are existed; the earliest right atrial activation is $\leq$15 ms preceding the P-wave onset or when AT has a monophasic positive P-wave in lead V1.[37,44] Earliest right atrial activation near the putative Bachmann's bundle region can also be seen in a left atrial origin.

Focal ATs can also arise from the epicardial structures, such as a ligament of Marshall. This ligament has been suggested to have multiple electrical connections with musculature of the left posterior free wall, near or inside the pulmonary vein ostium, or coronary sinus.[58] If the earliest endocardial activation site locates near the left pulmonary vein ostium or posterolateral portion of mitral annulus, a potential epicardial tachycardia focus need to be considered. For the ectopy from the ligament of Marshall, the earliest Marshall potential would be the ablation target site. Therefore, the differentiation of Marshall potential from the pulmonary vein or left posterior free wall potential would be necessary step. Differential pacing or direct recording techniques of the ligament of Marshall potential using the microelectrode catheter inserted into the vein of Marshall could distinguish the ligament of Marshall potential from the pulmonary vein potential to avoid misinterpretation and inappropriate RF energy application.[58]

After the earliest activation site is identified, detailed interpretation of the local electrogram can be useful to fine tune for localization of the targeted site. At the earliest activation site, the unipolar electrogram (unfiltered or high pass filtered at a low frequency such as 0.5 Hz) should have a QS morphology with an initial rapid intrinsic deflection (**Figure 6**).[8] Fractionated or spiked potentials are often present as a pre-potential at the successful ablation site, but may also be observed at other nonspecific sites.[11] Recently, De Groot et al.

reported that significant differences in electrogram fractionation, fractionation duration, and peak-to-peak voltage between AT origin and reminder area exist.[59] We also demonstrated that around 80% of ATs originating from low voltage zone (LVZ) or border zone around the LVZ.[60] The electrograms inside the LVZ demonstrated wide, low-amplitude, and fractionated electrograms, suggesting a delayed and nonuniform anisotropic conduction through the diseased atrium. This may be related to atrial fibrosis resulting from proliferation of smooth muscle cells and collagen fibers beneath the endocardial lining.[61] The mitral or tricuspid annulus ATs have large V and smaller A deflections recorded at their origin (**Figure 7**).

Paced Activation Sequence Mapping

Paced activation sequence mapping also can be useful technique to localize an AT origin.[5,10,12,14] Due to the limited spatial resolution of this technique, it is usually performed only as a guide of approximate focus location when AT is difficult to induce or nonsustained. Pacing is performed from the mapping catheter that is positioned at the area expected as the earliest activation site. Comparison of the P-wave morphology and endocardial activation sequence between pacing and AT beats can suggest whether the pacing site locates nearby or remote from the AT origin.

Images for Assessing Anatomic Structures

Selective angiography of SVC, coronary sinus, and pulmonary veins would help to evaluate the anatomic relationship between atrium and thoracic veins.[3,5,7,14,17] Balloon-occluded coronary sinus angiography with appropriate fluoroscopic projections would also help to visualize the vein of Marshall. Integration of three-dimensional (3-D) MRI or CT image with the left atrium/pulmonary vein geometry would be ideal way to identify the precise ectopic focus. Intracardiac echocardiography has been used in conjunction with conventional mapping catheter to assess the relation of mapping sites to endocardial structures and assess catheter stability for ablation. It is also useful for assessing the anatomical proximity of the SVC, upper portion of the crista terminalis, and right superior pulmonary vein, facilitating precise mapping and the interpretation of far-field potentials from neighboring structures.[11,32]

Three-Dimensional Contact and Noncontact Mapping

For catheter ablation of focal ATs, 3-D mapping systems have many useful functions that can create endocardial geometry, activation map, and voltage map. Furthermore, these systems can show real time movement of electrode tips for catheter navigation and can label the RF lesions and also interesting sites on the constructed endocardial geometry.

The CARTO system (Biosense Webster) is based on a sequential mapping technology that can reconstruct the cardiac chamber and activation sequence sampled using a roving catheter through point-by-point manner.[15,64] Repeated sampling from closely adjacent points can create high-resolution mapping in the interesting region for detailed mapping of the AT origin. The major limitation of this system is the requirement of point-by-point sampling so that it is unable to map the nonsustained ATs.

The EnSite system (Endocardial Solution) has been demonstrated to facilitate reconstruction of precise geometry, identification of ectopic foci, and navigation of catheter to the target sites.[60,65] Major advantage of this system is the simultaneous acquisition of more than 3000 unipolar electrograms from whole chamber, facilitating mapping of nonsustained AT, even in single beat.

RF Catheter Ablation

Ablation Techniques

After the AT focus is identified, RF energy with power (20 and 50 Watts) is delivered for 30 to 60 seconds. Rapid termination of AT within 10 seconds just after starting RF energy application is also a good marker of acute success. Heating effect by RF energy causes automaticity followed by quiescence with permanent tissue injury. Therefore, acceleration of AT followed by termination is also a preferable phenomenon that the RF energy is effective.

When we choose noncontact mapping guide ablation for focal AT, we ablate the AT origin and/or tissue along the proximal area of preferential conduction to eliminate baseline and also shifting AT foci.[60] Electrical isolation of the SVC can cure tachycardias originating from the SVC muscle sleeve despite continued focal electrical activity inside the sleeve.[40] Ablation of pulmonary vein tachycardia that targets the ectopic focus is highly effective. However, electrical disconnection of the arrhythmogenic pulmonary vein at the atria – pulmonary vein junction may be a preferable approach in some cases to avoid a risk of pulmonary vein stenosis.[43] Sanders et al. reported the ablation to electrical connection of the muscular coat surrounding the coronary sinus could allow to isolate an AT trigger of atrial fibrillation.[66] Electrical isolation of the coronary

Table 1 Outcomes of radiofrequency catheter ablation of focal atrial tachycardias.

Authors	Mapping Tool	Patient Number	Total Ablated AT Foci (R/L)	Multiple Foci	Success	Complications	Recurrence	Follow-up (Mo)
Walsh et al. (4) 1992	C	12	12 (5/7)	0 (0%)	11 (92%)	1 (8%)	1 (9%)	13 (range 3–21)
Goldberger et al. (17) 1993	C	15	15 (15/0)	2 (13%)	12 (80%)	0 (0%)	2 (17%)	19 ± 7
Tracy et al. (5) 1993	C	10	10 (8/2)	2 (20%)	7 (70%)	0 (0%)	2 (29%)	7 ± 4
Kay et al. (6) 1993	C	15	15 (14/1)	1 (7%)	15 (100%)	1 (7%)	3 (20%)	9 ± 4
Lesh et al. (7) 1994	C	17	22 (17/5)	NA	16 (94%)	0 (0%)	2 (13%)	10 ± 1
Wang et al. (9) 1995	C	13	15 (10/5)	1 (8%)	9 (69%)	1 (8%)	1 (8%)	17 ± 14
Poty et al. (8) 1996	C	36	36 (33/3)	3 (8%)	31 (86%)	0 (0%)	4 (13%)	18 ± 15
Pappone et al. (10) 1996	C	45	45 (36/9)	NA	42 (93%)	3 (7%)	3 (7%)	22 ± 12
Kalman et al. (11) 1998	C/ICE	27	31 (27/4)	NA	25 (93%)	0 (0%)	4 (16%)	10 ± 6
Chen et al. (67) 1998	C	112	105 (95/10)	17 (15%)	100 (98%)	0 (0%)	3 (3%)	NA
Weiss et al. (14) 1998	C	48	52 (40/12)	NA	44 (92%)	0 (0%)	5 (11%)	range 4–58
Natale et al. (15) 1998	CARTO	24	29 (16/13)	1 (4%)	24 (100%)	NA	1 (4%)	NA
Schmitt et al. (13) 1999	Basket	31/16*	31 (21/10)	3 (10%)	15 (94%)	NA	NA	NA
Weiss et al. (62) 2000	CARTO	15	15 (14/1)	0 (0%)	15 (100%)	0 (0%)	2 (13%)	9 ± 6
Anguera et al. (16) 2001	C	105	105 (91/14)	7 (7%)	80 (76%)	2 (2%)	8 (10%)	33 ± 15
Schmitt et al. (65) 2001	EnSite	10/8*	8 (8/0)	0 (0%)	7 (88%)	0 (0%)	0 (0%)	6 ± 2
Wetzel et al. (64) 2002	CARTO	32/30*	34 (22/12)	3 (10%)	33 (97%)!	0 (0%)	1 (3%)	16 ± 9
Homann et al. (63) 2002	C/CARTO	42/37**	38 (27/11)	3 (8%)	32 (86%)	0 (0%)	4 (13%)	7 ± 6
Higa et al. (60) 2004	EnSite	13	14 (14/0)	1 (8%)	12 (92%)	0 (0%)	1 (8%)	8 ± 5
Kistler et al. (54) 2006	C	186	196 (144/52)	NA	177 (90%)	NA	NA	NA
Total		808/784	828 (657/171)	44 (7%)	707 (90%)	8 (1%)	47 (8%)	

Number in parenthesis indicates reference number; AT = atrial tachycardia; Basket = Basket catheter guided; C = conventional mapping; CARTO = CARTO electromagnetic mapping system; EnSite = EnSite noncontact mapping system; ICE = intracardiac echocardiography; R/L = right/left sided AT; NA = data not available.
*: number indicates patients received ablation of AT;
**: only 37 patients completed the electroanatomic mapping and 38 out of 45 ATs received ablation;
!: numbers indicate number of AT foci, 33 out of 34 AT foci were eliminated in this study.
(modified from reference 74, Higa et al.)

Table 2 Outcomes of radiofrequency catheter ablation of septal atrial tachycardias.

Authors Published Year	Mapping Tool	Patient Number	Location of AT Origin	Total Ablated AT Foci (R/L)	Multiple Foci	Success	Complications	Recurrence	Follow-up (Mo)
Connors et al. (35) 2000	C	8	Koch triangle	8 (8/0)	0 (0%)	8 (100%)	1 (13%)	1 (13%)	NA
Frey et al. (37) 2001	C	16	Septum	16 (10/6)	0 (0%)	16 (100%)	0 (0%)	2 (13%)	8 (1–68)
Marrouche et al. (45) 2002	C/CARTO	5	Septum	5 (2/3)	0 (0%)	5 (100%)	0 (0%)	0 (0%)	14 ± 8

Number in parenthesis indicates reference number; AT = atrial tachycardia; C = conventional mapping; CARTO = CARTO electromagnetic mapping system; NA = data not available; R/L = right/left sided AT.
(adapted from reference 74, Higa et al.)

Table 3 Outcomes of radiofrequency catheter ablation of annular atrial tachycardia.

Authors Published Year	Mapping Tool	Patient Number	Location of AT Origin	Total Ablated AT Foci (R/L)	Multiple Foci	Success	Complications	Recurrence	Follow-up (Mo)
Morton et al. (34) 2001	C	9	TA	9 (9/0)	0 (0%)	9 (100%)	0 (0%)	1 (11%)	9 ± 6
Matsuoka et al. (38) 2002	C	5	TA/MA	6 (4/2)	1 (20%)	5 (100%)	0 (0%)	0 (0%)	32 ± 11
Kistler et al. (50) 2003	C	7	MA	7 (0/7)	0 (0%)	7 (100%)	0 (0%)	1 (14%)	17 ± 10
Gonzalez et al. (51) 2004	CARTO/NavX/C	10	MA-Ao J	10 (0/10)	0 (0%)	10 (100%)	0 (0%)	0 (0%)	24 ± 19

Number in parenthesis indicates reference number; AT = atrial tachycardia; C = conventional mapping; CARTO = CARTO electroanatomical mapping; MA = mitral annulus; MA-Ao J = mitral annulus-aortic junction; NavX = NavX navigation system; R/L = right/left sided AT; TA: tricuspid annulus.
(modified from reference 74, Higa et al.)

Table 4 Outcomes of radiofrequency catheter ablation of atrial tachycardias from thoracic veins (PV, CS, SVC, Marshall's ligament).

Authors Published Year	Mapping Tool	Patient Number	Location of AT Origin	Total Ablated AT Foci (R/L)	Multiple Foci	Success	Complications	Recurrence	Follow-up (Mo)
Kistler et al. (43) 2003	C	27	PV	28 (0/28)*	1 (4%)	28 (100%)	0 (0%)	4 (14%)	25 ± 22
Volkmer et al. (46) 2002	CARTO	1	CS	1 (0/1)	0 (0%)	1 (100%)	0 (0%)	0 (0%)	5
Navarrete (47) 2003	CARTO	1	CS	1 (0/1)	0 (0%)	1 (100%)	0 (0%)	0 (0%)	6
Kistler (42) 2005	C	13	CS ostium	13 (13/0)	0 (0%)	11 (85%)	0 (0%)	1 (9%)	25 ± 4
Kistler (48) 2005	C/CARTO	8	CS body	8 (0/8)	0 (0%)	8 (100%)	0 (0%)	0 (0%)	37 ± 13
Ino et al. (40) 2000	C	1	SVC	1 (1/0)	0 (0%)	1 (100%)	1 (100%)**	0 (0%)	15
Dong et al. (41) 2002	CARTO	1	SVC	1 (1/0)	0 (0%)	1 (100%)	0 (0%)	0 (0%)	3
Polymeropoulos et al. (49) 2002	CARTO	1	ML	1 (0/1)	0 (0%)	1 (100%)	NA	0 (0%)	3

Number in parenthesis indicates reference number; AT = atrial tachycardia; C = conventional mapping; CARTO = CARTO electromagnetic mapping system; CS = coronary sinus; ML: Marshall's ligament; PV = pulmonary vein; R/L = right/left sided AT; SVC: superior vena cava.
*: 25 ATs received focal ablation and 3 ATs received isolation of arrhythmogenic pulmonary vein;
**: transient phrenic nerve palsy.
(modified from reference 74, Higa et al.)

Table 5 Outcomes of radiofrequency catheter ablation and modification of sinus tachycardias.

Authors Published Year	Mapping Tool	Patient Number	End Points	Success	Complications	Recurrence	Follow-up (Mo)
Sinus Node Reentrant Tachycardia							
Sanders et al. (30) 1994	C	10	—	10 (100%)	0 (0%)	0 (0%)	9 ± 6
Inappropriate Sinus Tachycardia							
Lee et al. (31) 1995	C/ICE	16	Total Abl/25% HR*	16 (100%)	2 (13%)	2 (17%)	7 ± 2
Callans et al. (32) 1999	C/ICE	10	30 bpm**	8 (80%)	0 (0%)	NA	NA
Man et al. (33) 2000	C	29	<90 bpm***	19 (66%)	2 (7%)	6 (27%)	32 ± 12

Number in parenthesis indicates reference number; Abl = ablation; AT = atrial tachycardia; bpm = beats per minutes; C = conventional mapping; HR = heart rate; ICE = intracardiac echocardiography; NA = data not available; R/L = right/left sided AT.
*: total ablation of sinus node and the more than 20% reduction in the baseline sinus rate;
**: abrupt decrease (more than 30 bpm) in the baseline sinus rate;
***: reduction of the baseline sinus rate to <90 bpm, and a 20% or greater reduction in sinus rate during isoproterenol infusion.
(adapted from reference 74, Higa et al.)

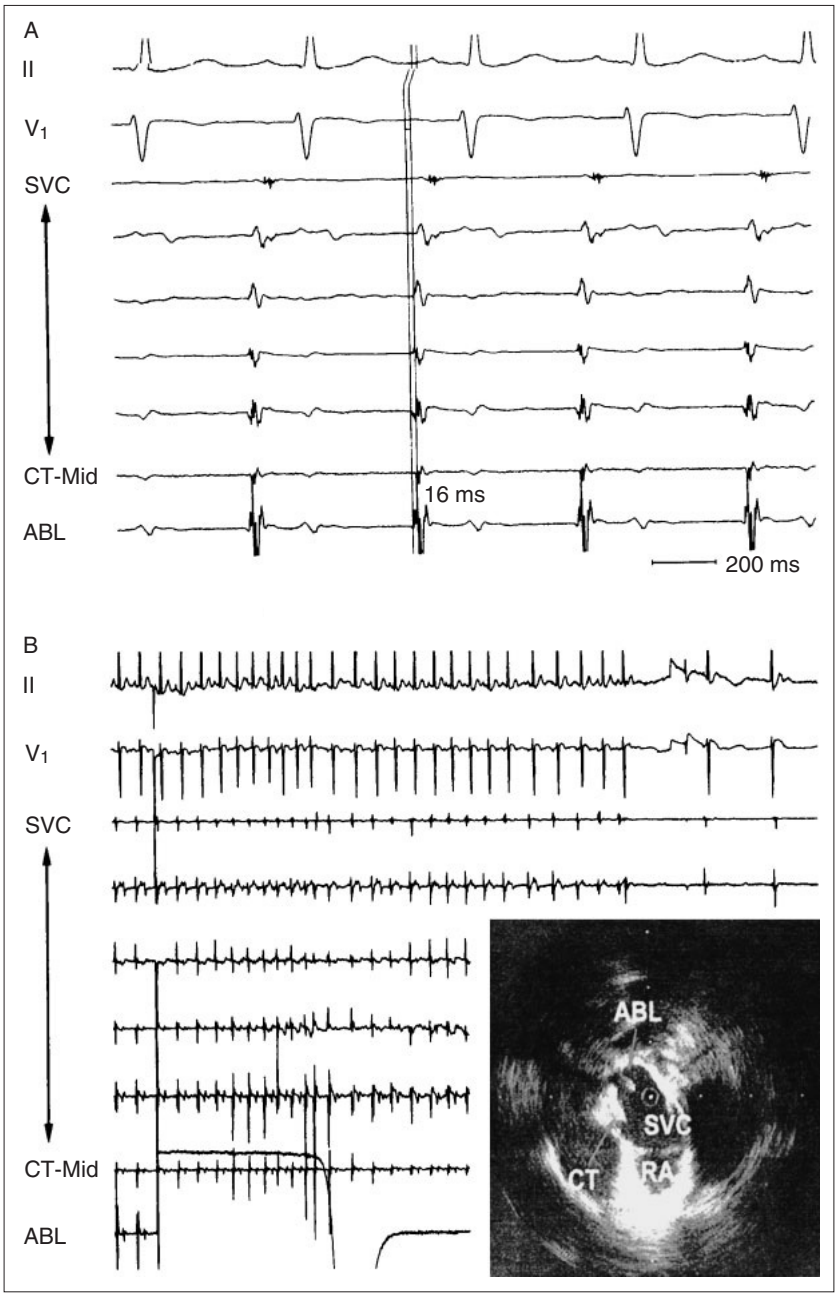

Figure 8 Modification of sinus node in patient with inappropriate sinus tachycardia.
Intracardiac echocardiography was used to locate the ablation catheter along the crista terminalis. Panel A: Intracardiac electrograms from the SVC and along the CT. The electrogram from the ablation site at the crista terminalis is 16 msec earlier than the P-wave. Panel B: RF ablation produces a sudden decrease of heart rate. Intracardiac echocardiography (inset in panel B) demonstrates the anatomical location of the ablation catheter tip (ABL), crista terminalis (CT), superior vena cava (SVC) and right atrium (RA). (adapted from reference 73, Hsieh and Chen).

sinus musculature may be an alternative approach when the focus-oriented approach can not cure AT from coronary sinus. If early activation site of AT arise from the area between the left superior pulmonary vein ostium and AV groove, AT from the ligament of Marshall is suspected. Triple discrete potentials are presented that can be distinguished from atrial and pulmonary vein potentials by pacing maneuvers to avoid inappropriate RF applications. Mechanical suppression of AT occurrence during catheter manipulation at the presumed AT origin may also be a good predictor of a successful ablation.[10] If mechanical trauma suppress AT for several hours, we may fail to further mapping and to assess the effectiveness of ablation lesions.

Efficacy and Safety

The outcomes in the catheter ablation of focal AT and sinus node modification in patients with IST have been reported from many institutes (**Table 1–5**). In the summary of a total of 808 patients with focal AT from 20 previous literatures, the average success

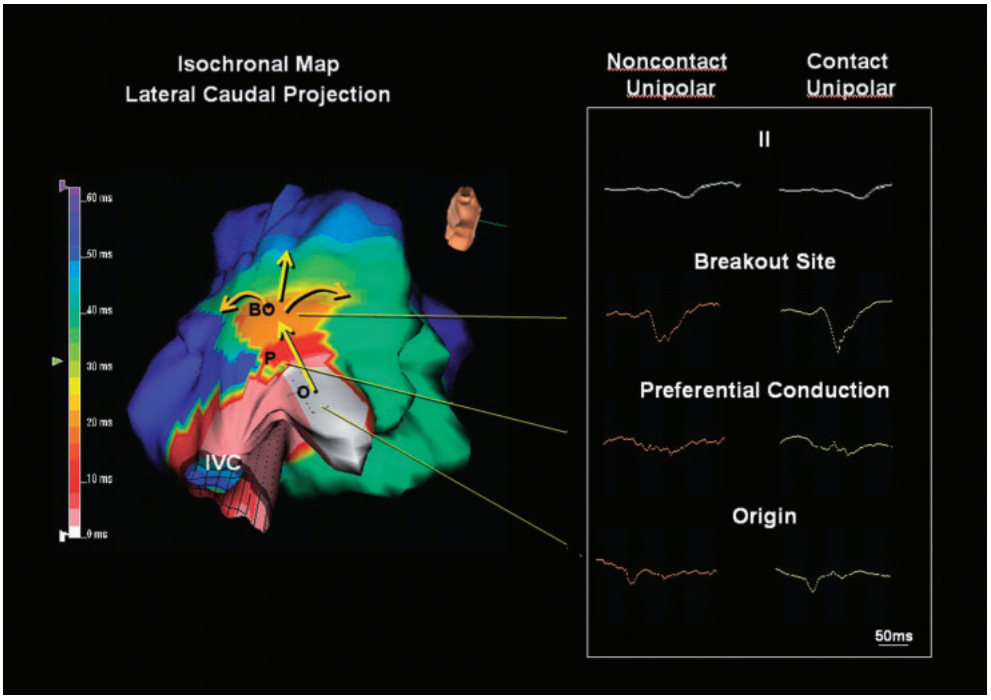

Figure 9
Left panel: Noncontact mapping of focal AT in the lateral caudal projection. Color scale for each isochronal map has been set so that white indicates earliest activation and purple indicates latest activation time. The focal activation originates from the anterior portion of RA-IVC junction and the wavefront propagates up to the middle portion of crista terminalis, and then spreads out to the whole atrium. Right panel: The contact and noncontact unipolar electrograms show a QS pattern at origin, and a rS pattern at breakout site. The contact and noncontact unipolar electrograms at the origin and proximal site of the preferential conduction reveal multi-components of electrogram deflections. Noncontact unipolar electrograms recorded at origin, preferential conduction, and breakout site were nearly identical with the contact electrograms obtained from these areas simultaneously.
O = origin of AT; P = preferential conduction; BO = breakout site of AT; IVC = inferior vena cava. (adapted from reference 60, Higa et al.)

and recurrence rate were around 90% and 8% (**Table 1**). In those summary data, ATs from left atrium was accounted for 20% and patients with multiple foci were seen in 7%. Success rate in the left-sided AT showed lower than in the right-sided AT. Patients with multifocal AT showed a higher recurrence rate than those with a single focal AT. Age is an independent risk factor for the existence of multiple ATs and the recurrence of AT after an initial successful ablation.[67] Complications of catheter ablation were observed in around 1% of patients including: cardiac perforation, phrenic nerve and sinus node injury.

Managements for Complications

If we need to ablate inside venous structures including pulmonary vein, SVC, and coronary sinus, limited temperature (less than 50–55 °C) can be safe to reduce the risk of thrombus formation, catheter adhesion to the venous wall, and venous stenosis.

For ablation of parahisian AT, lower RF energy and temperature settings (5–10 W increments starting from 10 W to a maximal output 40 W) and continuous monitoring of the AV conduction can be safe for avoiding AV block. Before applying a permanent cryoablation lesion (less than −70 °C), the use of cryomapping (−30 °C) to identify AT foci close to the AV node with monitoring the AV conduction is safe ablation procedure.[68]

If AT origin is suspected as an epicardial focus, a large-tip (8 mm) electrode catheter or saline-irrigated catheter may be useful for making deep lesion. Atrio-esophageal fistulas as a fatal complication can occur after aggressive radiofrequency ablation at the posterior aspect of the left atrium.[69] It is indispensable step to evaluate the anatomical location of the esophagus and to limit RF power when we ablate posterior wall of left atrium.

Sinus node modification for inappropriate sinus tachycardia often needs extensive catheter ablation

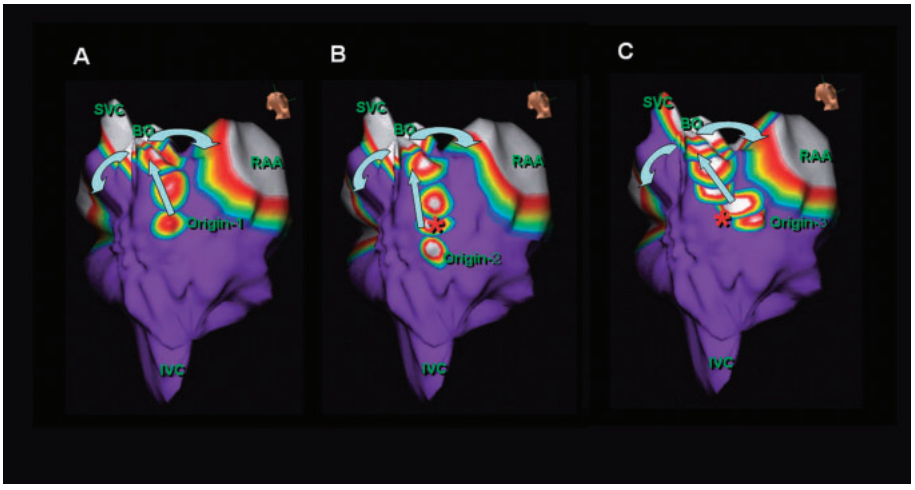

Figure 10 Noncontact mapping of focal AT in the posterolateral view.
Isopotential maps show overlapping of the activation wavefront located at origin, preferential conduction, and breakout site of baseline beat (Panel A), and adenosine-induced shifting beats (Panel B and C). In the baseline beat, the focal activation originates from the middle portion of crista terminalis, the wavefront propagates up to the posterior portion of RA-SVC junction through the preferential conduction area, then reaches the breakout site, and spreads to the whole atrium (Panel A). Panel B: Adenosine (3 mg)-induced maximum shifting of AT origin in the caudal direction. Panel C: Adenosine (6 mg)-induced maximum shifting of AT origin in the rightward direction.
$*$ = baseline origin of AT; SVC = superior vena cava; RAA = right atrial appendage. (adapted from reference 72, Higa et al.)

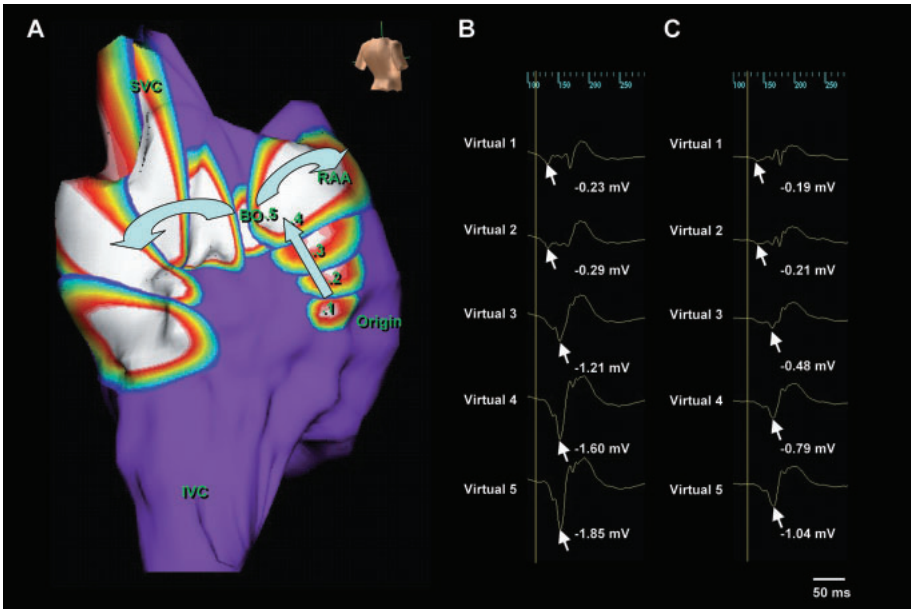

Figure 11 Noncontact mapping of focal AT in the posterolateral view.
Panel A: In the baseline beat, the focal activation originates from the middle lateral right atrium, the wavefront propagates up to the high lateral right atrium through the preferential conduction area, then reaches the breakout site, and spreads to the whole atrium. Panel B: Virtual electrograms at origin (Virtual 1), proximal (Virtual 2), mid (Virtual 3), distal (Virtual 4) portion of preferential conduction, and breakout site (Virtual 5) in the baseline period. Panel C: Virtual electrograms (Virtual 1–5) of last beat from adenosine (6 mg)-induced termination of AT. Arrows indicate first peak negative deflections of each locations. The value (mV) with an arrow indicates the peak negative voltage of each location. The last beat just before termination showed decrease in the peak negative voltage at origin, preferential conduction, and breakout site.
BO = breakout site of AT; IVC = inferior vena cava; Origin = baseline origin of AT; SVC = superior vena cava; RAA = right atrial appendage. (adapted from reference 72, Higa et al.)

(**Figure 8**). RF applications should initially apply from the most superior portion of the crista terminalis, with subsequent lesions to the inferiorly along the crista terminalis to achieve approximately a 25–30% decrease in the maximal heart rate during the isoproterenol and/or atropine loading. Several complications can result from aggressive ablation in the sinus node region.[31–33] Acute or late occurrence of SVC syndrome has been reported. Sick sinus syndrome after ablation will need a pacemaker implantation. Furthermore, right phrenic nerve injury can occur. To avoid phrenic nerve injury, diaphragm twitching by high output pacing from ablation catheter prior to RF application can be useful.

New Insights from Recent Studies

This laboratory reported that noncontact mapping demonstrated a non-radial activation pattern of focal AT (**Figure 9**).[60] Focal AT originates from a small area and spreads out to the whole atrium through a preferential conduction, non-uniform and not radial activation pattern. We also demonstrated that the global voltage analysis using unipolar peak negative potential and evaluated distribution of LVZ and the voltage at AT origin was significantly lower than that of preferential conduction and AT breakout site.[60] Based on the novel findings from these two studies, we have proposed the concept of atrial myopathy in patients with focal AT, and most of the focal AT origin and preferential conduction were inside the LVZ or border zone around the LVZ. Thus, the atrial myopathy is characterized by the structural and functional changes in atrial substrate which is related to the locations of AT origin and breakout site. Sanders et al. also showed non-radial activation pattern of focal AT evaluated by high-density contact mapping catheter.[57]

Information about relationship between substrate property and AT origins is limited. Previous studies on the atrial specimen resected from the area with atrial arrhythmias showed a slow response action potential with spontaneous depolarization.[24] Josephson et al. also showed the slow response or depressed fast response action potential from the atrial specimen resected from human AT.[25] These findings may support our proposed hypothesis that focal AT originates from diseased atria and explain the possible mechanism of ATs originating from LVZ or border zone around the LVZ.

The noncontact unipolar electrograms in the LVZ demonstrated wide, low-amplitude, and fractionated electrograms suggesting a delayed and nonuniform anisotropic conduction through the diseased right atrium. This may be related to atrial fibrosis resulting from proliferation of smooth muscle cells and collagen fibers beneath the endocardial lining.[61] Recently, De Groot et al. demonstrated fragmented, long-duration, low-amplitude electrograms characterized the origin of focal AT.[59] They showed the different characteristics in fractionation, fractionation duration, and amplitude of atrial potentials between AT focus and remainder of the atria. They suggests that the severity of fractionation probably reflects the extent of interstitial fibrosis and can influence wavefront conduction, and higher extent of fractionation means that poor cell-to-cell coupling at the AT origin exist. Impaired cell-to-cell coupling may induce conduction abnormality suggesting possible underlying mechanism of reentry.

Possible Roles of Preferential Conduction

Previous studies demonstrated electrical impulses from the sinus node region could not conduct to the atrium when conduction in the zone of perinodal fibers becomes depressed due to pathological conditions.[70] Furthermore, the concepts from previous studies may explain that ablation of specific fiber connected from the ectopic origin can create the exit block from origin.[71] Although this conduction may not be electrically protected either permanently or functionally in almost cases, the preferential conduction may play a critical role in ablation of focal AT in some cases. In such cases, we can choose the ablation target according to the concept of origin and preferential conduction of AT. Recently, we evaluated the adenosine-induced termination of focal AT using high-resolution mapping.[72] In this report, we first demonstrated the shifting of the AT origin in 50% of adenosine-induced termination episodes with higher extent of cycle length variation compared to baseline AT, and adenosine significantly decreased the peak negative voltages at origin, preferential conduction, and breakout site before AT termination (**Figure 10** and **11**). In this study, we also showed that applications of RF energy on the origin and/or proximal portion of preferential conduction could eliminate baseline and shifting AT foci. These findings suggest that the focal AT can arise from some extent of arrhythmogenic area, not the single discrete focus, and can shift subsidiary AT foci after ablation of dominant foci.

Although the non-radial activation pattern of focal AT may mimic macroreentrant AT, well understanding the electrophysiological property and concept of AT origin with preferential conduction can help to avoid misdiagnosis and inappropriate ablation.

Conclusions

Diagnosis of focal AT can be made by an electrophysiologic study, and localized by standard endocardial mapping and/or 3-D mapping systems. Information about anatomic–electrophysiologic relationships is often useful for guiding ablation of focal AT. Almost focal ATs can be cured with a high success rate, low recurrence and low complication rate when appropriate attentions are taken.

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